Initial Comments and Notes on "Appendix G, Baseline Ecological Risk Assessment", DRAFT, Portland Harbor RI. Prepared for The Lower Willamette Group by Windward Environmental, Dated August 19, 2009.

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BIG PICTURE ISSUES:

1. Treatment of dioxin and furan risk:
    a. *Surface Water, TZW, Sediment and Invertebrate Tissue:* Important and prevalent congeners are dropped in surface water, transition zone water, sediment and invertebrate tissue due to "no SLV/TRV". Where appropriate, TRVs can be derived using TEFs (e.g. fish exposure to sediment, TZ water and surface water) and an appropriate 2,3,7,8-TCDD. For aquatic biota besides fish for which TEFs are not available, Total dioxin / furan concentration (sum of 12 congeners) should be compared to the 2,3,7,8-TCDD SLV. It is defensible to assume that toxicity in this case would be similar to TCDD. In either case, the risk assessment needs to acknowledge that there are other dioxin and furan risk drivers besides 2,3,7,8-TCDD and these should be carried through the screening process. Concentrations for these congeners should be presented in the risk screening.
    b. *Fish Tissue:* Risk is underestimated do to the use of TRV that is not defensible. Based on a literature review, there is a high probability of risk to fish at localized areas of the site.
    c. *Wildlife Risk:* Risk to wildlife is presented as a "total dioxin TEQ risk" or a "total TEQ". Congener specific contribution to TEQ risk is not presented and does not lay the groundwork for appropriate PRG selection. Instead, congener drivers for bird risk are selected to represent mammalian risk and vice versa, resulting in higher acceptable risk levels than would be calculated by looking at each individually.
        i. Osprey: Risk is underestimated as the bird egg line of evidence is dropped, and the dietary TRV does not incorporate uncertainty. Proper evaluation of these two lines of evidence shows that Dioxin / Furan TRV (also applies to PCB TEQ, dioxin / furan TEQ, PCB TEQ) and associated Threshold Tissue Concentrations. The TRV is based on the Noseck paper looking at pheasant dietary exposure to TCDD. DEQ uses the same paper in Guidance, but follows EPA's lead from the Great Lakes in including an uncertainty factor of 10 based on that fact that the NOAEL resulted from a 10-week exposure, which would have achieved only 13 percent of steady-state accumulation. They concluded that an order of magnitude lower concentration in the food could still have elicited the same tissue levels and effects (U.S. EPA 1993).

The difference in this interpretation results in a TRV LOAEL TRV of 7.0 E-6 mg/kg/day (Guidance for Assessing Bioaccumulative Chemicals of Concern in Sediment) based on the Great Lakes work (DEQ mutliplies the NOAEL x 5 to estimate the LOAEL; however the LOAEL of 1.4 E-5 could also be used incorporating the UF). This dietary TRV is also used by the RSET in bioaccumulation chapter to determine fish tissue and sediment acceptable levels. If this TRV is used instead of the LWG TRV the acceptable fish tissue level (or TSC in this document) goes from 665 ng/kg fish tissue to from 33.3 to 79 ng/kg fish tissue LOAEL) and 2.3 to 8 ng/kg (NOAEL). This dietary TRV is also more in line with the egg based TRVs (2.3E-6 LWG based on chicken; 4.0 E-4 DEQ based on pisc. birds) and corresponding acceptable fish tissue concentrations ranging from 3.2 (LWG) to 40 ng/kg (DEQ) fish tissue for protection of bird eggs (see comment on chicken TRV for bird egg). The use of a more relevant TRV puts the both bird lines of evidence on the same scale, and results in similar risk determination (it should be noted that egg based and dietary based LOAEL values DEQ uses are the same - 40 ng/kg ATL in fish). Based on multiple lines of evidence, the acceptable fish tissue concentration for the protection of birds should be between 40 ng/kg (egg) to 79 ng/kg(dietary) instead of the LWG's calculated TSC of 665 ng/kg. This will change the risk analysis - namely dioxin/furan TEQ, PCB TEQ and Total TEQ would screen in for the dietary pathway (Max tissue concentration 232 ng/kg dioxin/furan TEQ; 196 PCB TEQ; 262 Total TEQ). Risk conclusions would be similar as for bird egg estimates (e.g. see Section 5.2 in Attachment 17. Interestingly, the use of 40 ng/kg as the acceptable level would also be more in line with a level protective of fish.

d. Sediment PRG: The back-calculated PRG for this congener for human health is close to the value presented in DEQ's bioaccumulation guidance. However, for birds especially, this value does not match up, most likely because of differences in the bird TRV. That means if an appropriate TRV is used as outlined above, the sediment PRG for birds would be about 3.5 E-6 mg/kg instead of the mammalian numbers that are currently presented of 2.61 E-5 and 1.71E-4. ***Bird PRGs need to be calculated because these will be the driver over ecological mammalian risk for 2,3,4,7,8-PCDF.***

   i. Additionally, PRGs should be calculated for 2,3,7,8-TCDF (birds) and 1,2,3,7,8-PeCDD (mammals)

2. Elimination of Chemicals: Chemicals eliminated due to not TRV or high detection limits were not carried through the risk assessment. See comments on SLERA and Refined Screen. In many cases, appropriate TRVs do exist.

3. Consideration of Localized Risk: Final risk characterization is based on site wide risk only. Receptor exposure scenarios outlined by EPA in the problem formulation are not followed, and risk characterization is based on scenarios proposed by the LWG in this document. The result is that site wide risk is emphasized, while localized risk is downplayed. As examples many contaminates showing an HQ>1 were dropped in fish, invertebrate, amphibian, and plants. PRGs were not developed for these compounds.

4. Key Lines of Evidence Dropped: Osprey Egg and Transition Zone water represent large lines of evidence that were dropped in the final characterization of the risk assessment.
    a. Section 6.7, Page 213 "TZW was evaluated but was not used to identify COCs and is therefore not discussed further in the conclusions".

5. Uncertainty Section: The uncertainty discussed is one-sided and does not present the true uncertainty that is evident throughout the document in how chemicals are dropped due to high detection limits, no TRVs, or uncertainty in the final selection of exposure and effect estimates.
6. Estimates of Exposure: The model is used extensively to predict concentrations of benthic invertebrates. How does the model matchup between the empirical data and the modeled data? How do BSAFs match up to the modeled data?
7. Didn't show work; process steps missing: I cannot find important pieces needed for review. One example is the 95% UCL concentrations used for all receptors and media.
8. Link to PRGs:
    a. PRGs are not based on the bird egg line of evidence
    b. Food Web Model:
        i. A site-wide food web model was used to back calculate to sediment. If we are protecting local breeding pairs of osprey, as was outlined in the problem formulation, then PRGs need to be based on the 1-mile exceedances seen in fish. This information needs to be used to back-calculate to sediment for PRGs. Instead, ***localized identified risk is then handled by a site-wide model that is then not protective of localized areas***. One example of this is RM 7 and dioxins and furans (dioxin TEQ or Total TEQ).
        ii. The use of surrogate dioxin and furan or PCB congeners to represent TEQ numbers. Instead of this methodology, congeners with identified risk in the risk assessment should be used to develop PRGs. The congeners that driver risk should be run in the food web model individually to ensure calibration and validation of predicted tissue concentrations due to differences in physical properties, and chemical distribution in sediment and tissue. Currently, PRGs for species that have more than one primary congener driving risk are represented only by the surrogate chemical (one each for PCB and dioixin /furan TEQ). For birds from about RM6.5 to 7.5 the main driver of TEQ is dioxin and furan risk, specifically two furan congeners. Is

the surrogate developed properly to endure these risks return to acceptable levels?

1. Bird dioxin TEQ: Driven primarily by 2,3,7,8-TCDF, 2,3,4,7,8-PCDF, 1,2,3,7,8-PCDD and 2,3,7,8-TCDD, respectively. But, the concentrations of these congeners vary in different tissue and sediment. ***These differences need to be evaluated by running the other congeners in the food web model.***

iii. ***This methodology does not address Total TEQ***, as the same process is not carried out to determine a surrogate; no regressions are presented in Table 7 (Early PRGs, March 2009). PRGs based on Total TEQ could be significantly different.

c. Site wide BSARs were used instead of BSAFs. There was nothing wrong with the methodology they used, only that it was artificially restrictive, leaving us with a lot of chemicals without BSARs or BSAFs. What was used as estimates when there was no modeled concentrations available (e.g. no food web model and no BSAR model available)?

1. **SLERA**

a. Surface Sediment SQGs need to be reviewed. There are numbers based on JSCS that should not be used as an early screen – several selected are severe effects thresholds or upper effects thresholds. In addition, several SQGs are based on Sediment Quality Standards (SQGs) from Ecology, 1995. These values are Sediment Management Standards for marine environments. These values are mostly toxicity based numbers, but chemicals for which there was not sediment SQG identified were dropped from further screening in tissue.

i. Comparison of maximum surface sediment COI concentrations to SQGs. This is a reduced list (see below) but a few COIs are eliminated – e.g. Antimony, Dibutyl phthalate, and two VOCs tetrachloroethene and trichloroethene.

ii. Table 2-3 shows surface sediment COIs with no SQGs. There are several important COIs on this list for which I believe appropriate sediment values can be obtained, or appropriate surrogates selected. This would include the butyltins, dioxins and furans (in addition to 2,3,7,8-TCDD), pesticides such as Lindane, Endrin ketone, mirex, toxaphene, herbicides, cyanide, perchlorate and many others. However, if sediment SQGs cannot be obtained for these chemicals, they should still be evaluated in the tissue screening step. ***It appears that once chemicals are screened out in this step they are not carried forward or shown in any tissue screen.*** The implications of this step are important as concentrations of these chemicals can be significant, but are not evaluated any further (see comments below).

b. Tissue Residue: Tissue residue benchmarks presented in Table 3-1, and I am assuming these have been reviewed by EPA. I only had time to do a minimal review of presented

values, but it is clear that some COIs are missing from this list. **This list should include all chemicals detected in tissue, and should not be a reduced list based on the sediment screen (chemicals without sediment SQGs - Table 2-3 - were not evaluated further in tissue screens). Also, it is unclear if there were COIs detected in fish tissue that were not detected in sediment.**

1. Invertebrate/ Dioxins and Furans: The 2,3,7,8-TCDD tissue residue value of 767 pg/g for invertebrates has not been reviewed. This value should be a "Total dioxin and furan" number, such that the sum of all 12 dioxins and furans should be screened against an appropriate dioxin SQG (no TEF used, just total concentration). A reasonable screening number for 2,3,7,8-TCDD might be 50 pg/g, which was calculated by Shephard (*Quantification of Ecological Risks to Aquatic Biota from Bioaccumulated Chemicals*).
2. Fish/Dioxins and furans: Dioxin fish tissue residue TRV is also not supportable, as it is based on fish egg to adult model that has a large amount of uncertainty associated with it. In absence of a species sensitivity distribution by EPA, the early protocol established of a BCF X AWQC should be used. This would come up with a value of about 40 pg/g. DEQ calculated a value from a species sensitivity distribution.
3. Di-n-octyl phalate: This value is much too high to be a screening level benchmark. Further review is necessary. This value is used to screen out a hit of 2,100 ug/kg ww in smallmouth bass.
4. Pesticides: DEQ has developed an extensive list for pesticides as a part of the Rhone Poulenc upland investigation. This may be a good starting point for review of this table.

ii. Comparison of maximum tissue concentrations to screening levels. This includes Table 3-5 (invertebrates), and Table 3-6 (fish).

1. Table 3-5 Invertebrates:
    a. Lab clam and worm concentrations have been adjusted to represent steady state conditions. More information is needed to ensure this adjustment was done appropriately. These concentrations form the basis for the exposure point concentrations for these species. I have obtained the reference they used and it appears incorrect – esp. in regards to PAHs. However, I need more time to review this piece. Presented in Attachment 4, BERA data, steady state estimates of invertebrate tissue needs to be reviewed.
    b. Dioxins and furans: The concentration is on 2,3,7,8-TCDD, and misses the evaluation of more prominent dioxin and furan congeners. Invertebrate concentrations are not presented for the other congeners. Table 3-5 shows the concentration of 2,3,7,8-TCDD to have the highest concentration in lab worm at

493 pg/g ww. However, concentrations of furan congeners are also very high such that a total dioxin furan concentration would well exceed the benchmark. Total dioxin furan concentrations are not presented here, but can be found in XXX of the document.

c. Other chemicals screened out in the **SLERA sediment "no SLV"** step (Table 2-3) have very high concentrations in tissue but **are not carried forward into the tissue screening steps**. These chemicals are also not identified in the benthic invertebrate tissue screening level benchmarks (Table 3-1) nor as chemicals without SLVs (Table 3-3). For example, max butyltin ion tissue concentration in worm tissue is 3,600 ppb (BT023) but this information is not presented in the invertebrate screening. Just because there is not a sediment toxicity SQG for the chemical does not mean it shouldn't be evaluated for tissue residue effects. This step will have to be revised.

d.

2. Table 2-6 Fish:
    a. Comparison of maximum detected concentration in fish should include all fish. For example, carp concentrations here are marked with a "NE" meaning not evaluated as a receptor in the ecological risk assessment. Screening all fish insures we are adequately protecting all species with our representative fish for each guild. This table does not provide enough information to do that assessment.
    b. Dioxin and furan congeners should screen in, and concentrations of the individual congeners should be presented in addition to 2,3,7,8-TCDD.

iii. Table 3-3 shows tissue COIs without SLVs for invertebrates and fish. Every effort should be made to locate appropriate values, as these are essentially dropped from the risk assessment. Presented below are some examples, but not the full review. This includes manganese, benzoic acid, benzyl alcohol, bis(2-chloroethoxy) methane, 4-chloro-3-methylphenol, 4-Nitorphenol, and nitrobenzene. These need to be reviewed, as several values have been developed by upland site efforts. Tables 3-5 and 3-6 really should have shown the maximum detections of these chemicals even though an SLV hadn't yet been identified.

1. Manganese: 2.2 mg/kg (US Army Corps of Eng. Residue Database).
2. 4-Chloro-3-methylphenol: 0.11 mg/kg (AWQC X BCF) – *Shephard PRE Table*

c. Fish Dietary: Table 4-3 shows the detection status of fish dietary COIs and Table 4-4 shows the dietary screening-level thresholds. These thresholds need to be reviewed – I could not complete this review. In addition, Table 4-5, which identifies "fish dietary COIs with no screening level threshold" needs to be reviewed. *PAHs do not seem correct, and many are let out in the "no SLV" category. *Need numbers for butyltins.

   i. *More details are presented on the Exposure and Effects Assumptions for the Fish Dietary LOE in Attachment 13, more information on fish dietary TRVs are presented in Attachment 14.*

   ii. Table 4-6, Maximum tissue (prey) and sediment concentrations compared to TTC or TSC. This isn't a great use of the "acceptable tissue" concept because it is really the combined DOSE of tissue and sediment that is necessary to give an HQ. Separating the tissue and the sediment is confusion and incorrect? The COIs without SLVs for this pathway need to be carried forward.

   iii. Each fish receptor is given a specific diet as outlined in Table 4-2. Due to the uncertainty in knowing what species different fish are feeding on, the Problem Formulation gave specific direction on how to move forward with the fish dietary evaluation. "Include realistic representations of sculpin or smallmouth bass home range (500 to ¼ mile on one side of the river). For sculpin and smallmouth bass, use a back calculation of the fish dietery risk equation to calculate an acceptable tissue concentration in prey for the protection of fish using the dietary equation, and acceptable dietary dose using EPOA direction on dietary TRVs." This analysis was to be specific to small home range fish and in doing so "will provide information about protection of larger home range omnivorous and insectivorous fish…". Acceptable tissue concentrations were to be calculation and applied to all benthic prey including (for both species) field and laboratory clams, lab worms, crayfish and sculpin. Instead, the bass evaluation is limited to worms, crayfish and sculpin. I would recommend simplifying the approach as was outline in EPA's problem formulation by appropriate TRV and evaluating **potential ranges in ingestion and body weight input parameters**, calculating acceptable tissue concentration, and using this information to screen all tissue. This information can be plotted on a map and clearly identify areas of concern for fish dietary risk. We do not know enough about fish dietary parameters to have confidence in parsing out specifics on ingestion parameters and body weights and sediment ingestion to have any confidence in this approach for individual fish. For body weight, this model should go beyond the "average of field collected data" used in this assessment and instead focus on the range in body weights expected in the field.

   iv. Table 4-1, Body and Food Ingestion Rates: These values will have to be reviewed and re-worked. The Gobas equation is flawed for this use because it is tied to the use of an "average of temperatures collected by ODEQ" and not a realistic assessment of potential ingestion. Previous equations in the Round 2 Report should be reviewed.

v. For the reasons stated above, I did not review Table 4-6 (largescale sucker), Table 4-7 (Juvenile White Sturgeon), Table 4-8 (Juvenile Chinook Salmon), Table 4-10, (Peamouth), and Table 4-12 (Pikeminnow). These tables take the risk analysis beyond what the method can do and determine risk for which we can have no certainly.

vi. Table 4-11 (Smallmouth Bass) and Table 4-9 (Sculpin) need to be re-worked as per EPA's problem formulation.

d. <u>Wildlife Dietary:</u>

i. Table 4-21: Body Weights and Ingestion Rates for Wildlife Receptors. These have changed since the Round 2 Report, so they will have to be reviewed again. Where ingestion rates are available, those should be used instead of generic equations from Nagy, 2001. This should be updated. Where are the references for these ingestion rates?

ii. Table 4-22: Prey for Wildlife Receptors are given here.

1. All diet "menu" is o.k. relative to the problem formulation except for Bald Eagle - smallmouth bass should be added to the dietary menu from which to pull the maximum tissue concentration.

iii. Table 4-23: Dioxins and furans should be listed out by congener.

iv. Table 4-24: Bird Dietary Screening Numbers (TRVs).

1. Table 8-11, Bird Dietary-Dose TRVs:

a. Dioxin / Furan TRV (also applies to PCB TEQ, dioxin / furan TEQ, PCB TEQ) and associated Threshold Tissue Concentrations. The TRV is based on the Noseck paper looking at pheasant dietary exposure to TCDD. DEQ uses the same paper in Guidance, but follows EPA's lead from the Great Lakes in including an uncertainty factor of 10 based on that fact that the NOAEL resulted from a 10-week exposure, which would have achieved only 13 percent of steady-state accumulation. They concluded that an order of magnitude lower concentration in the food could still have elicited the same tissue levels and effects (U.S. EPA 1993). The difference in this interpretation results in a TRV LOAEL TRV of 7.0 E-6 mg/kg/day (Guidance for Assessing Bioaccumulative Chemicals of Concern in Sediment) based on the Great Lakes work (DEQ multiplies the NOAEL x 5 to estimate the LOAEL; however the LOAEL of 1.4 E-5 could also be used incorporating the UF). This dietary TRV is also used by the RSET in bioaccumulation chapter to determine fish tissue and sediment acceptable levels. If this TRV is used instead of the LWG TRV the acceptable fish tissue level (or TSC in this document) goes from 665 ng/kg fish tissue to from 33.3 to 79 ng/kg fish tissue LOAEL) and 2.3 to 8 ng/kg (NOAEL). This

dietary TRV is also more in line with the egg based TRVs (2.3E-6 LWG based on chicken; 4.0 E-4 DEQ based on pisc. birds) and corresponding fish tissue concentrations ranging from 3.2 (LWG) to 40 ng/kg (DEQ) fish tissue for protection of bird eggs (see comment on chicken TRV for bird egg). The use of a more relevant TRV puts the both bird lines of evidence on the same scale, and results in similar risk determination (it should be noted that egg based and dietary based LOAEL values DEQ uses are the same - 40 ng/kg ATL in fish). ***Based on multiple lines of evidence, the acceptable fish tissue concentration for the protection of birds should be between 40 ng/kg (egg) to 79 ng/kg(dietary) instead of the LWG's calculated TSC of 665 ng/kg.*** This will change the risk analysis - namely dioxin/furan TEQ, PCB TEQ and Total TEQ would screen in for the dietary pathway (Max tissue concentration 232 ng/kg dioxin/furan TEQ; 196 PCB TEQ; 262 Total TEQ). Risk conclusions would be similar as for bird egg estimates (e.g. see Section 5.2 in Attachment 17.

v. *More information on recommended literature-based wildlife TRVs are presented in Attachment 14.*

vi. Table 4-15: Mammalian Dietary Screening Thresholds. THESE NEED TO BE REVIEWED.
    1. For Chordane isomers (Cis-Chlordane, trans-Chlordane, oxychlordane, trans-nonachlor, cis-nonachlor and Total Chlordane) the TRV of 0.24 mg/kg day, with a corresponding TTC of 183 ug/kg should be used. Total Chlordane should include the total of the above isomers.
    2. Dieldrin – the TRV should be 0.0077 mg/kg and the TTC 7 ug/kg. This will change the screening conclusions.
    3. Lindane isomers (should be presented as total hexachlorocyclohexanes) TRV should be 0.57 mg/kg day and the TTC 479 ug/kg.
    4. NEED TBT review!
    5. I have comments on the others, but no time to finish. I will send along DEQ's spreadsheet for review.

vii. Table 4-26: Dietary COIs with No Bird or Mammal Dietary Screening-Level Thresholds. ***Needs to be reviewed – add Rhone P review.
    1. Silver – Both a mammalian and Avian number is available from Eco SSL document (30.1 mg/kg day mammal and 2.02 mg/kg day bird).
    2. Manganese – Also values available from Eco SSL document (258 mg/kg day mammal and 179 mg/kg day bird).
    3. These are two examples, but the rest in this Table should be checked in detail – look at DEQ's table as a place to start.

viii. Tables 4-27 through 4-32 – Comparison of Maximum prey concentration to threshold concentrations for wildlife receptors. This needs to be reviewed after we agree on ingestion / body weights and TRVs.

e. <u>Bird Egg:</u>
   i. Bird egg Screening Level Thresholds are presented in Table 4-40. I HAVE COMMENTS ON THIS
   ii. Selected BMFs used to estimate bird egg tissue residues are presented in Table 4-41. CHECK BMFs

f. <u>Surface Water:</u> Surface water COIs are presented in Table 5-1 and TRVs in Table 5-2. Table 5-2 along with Table 5-3 (COIs with No Chronic TRVs) is going to need significant review. There are several COIs in Table 5-3 for which TRVs should be derived. See also Attachment 10 "Selection of Water TRVs" for more information.
   i. Table 5-3, Surface Water TRVs: (these are examples and do not represent a complete review.
      1. Dioxin (2,3,7,8-TCDD): The value reported here as 100 pg/L is incorrect. It should be 10 pg/L (AWQC).
      2. PCBs: The value for the Aroclors should be 0.014 ug/L, and the total of any group still needs to be less than 0.014 ug/L.
      3. NOT DONE WITH REVIEW
   ii. Table 5-3, Surface Water COIs with No Chronic TRVs. These are examples only and not full comments. ***DEQ has compiled an extensive list of water (and tissue) TRVs as a part of the Rhone Poulenc site.*** Perhaps this would be a good place to start.
      1. Dioxins and furans: I would argue that water TRVs for protection of fish can be defensible calculated using TEFs and the appropriate water SLV for 2,3,7,8-TCDD. For aquatic biota besides fish for which TEFs are not available, Total dioxin / furan concentration (sum of 12 congeners) should be compared to the 2,3,7,8-TCDD SLV. It is defensible to assume that toxicity in this case would be similar to TCDD. In either case, the risk assessment needs to acknowledge that there are other dioxin and furan risk drivers besides 2,3,7,8-TCDD and these should be carried through the screening process. Concentrations for these congeners should be presented in the risk screening.
      2. MCPP: There is a value for MCPA that can be used as a surrogate: 2.6 ug/L from the Canadian Water Quality Screening Values.
      3. 2,4-DB: A value of 4 ug/L is available, also using Canadian Water Quality Benchmarks.
   iii. Table 5-4 , Comparison of Maximum Surface Water Concentration to Chronic TRVs.

1. Are the metal surface water concentrations expressed only as dissolved concentrations? What about total concentration? How was dissolved calculated when the filter was used since they were using a large filter size (.5 um)?? This doesn't seem to fit a dissolved concentration (definition)?

g. Transition Zone Water:
   i. Table 6-1 lists the TZ water COIs
   ii. Table 6-2 lists the water TRVs for TZW COIs: These need to be reviewed. Instead of citing ODEQ, the original source should be cited. There seem to be more citations of source control numbers which may not be appropriate chronic TRVs. LARGE MASTER LIST OF WATER TRVs NEEDED
   iii. Table 6-3, TZW COIs without screening-level benchmarks: This table needs to be updated with TRVs. I would argue that water TRVs for protection of fish can be defensible calculated using TEFs and the appropriate water SLV for 2,3,7,8-TCDD. For aquatic biota besides fish for which TEFs are not available, Total dioxin / furan concentration (sum of 12 congeners) should be compared to the 2,3,7,8-TCDD SLV. It is defensible to assume that toxicity in this case would be similar to TCDD. In either case, the risk assessment needs to acknowledge that there are other dioxin and furan risk drivers besides 2,3,7,8-TCDD and these should be carried through the screening process. Concentrations for these congeners should be presented in the risk screening.
   iv. Are the metal surface water concentrations expressed only as dissolved concentrations? What about total concentration?

2. **REFINED SCREEN (ATTATCHMENT 5):** Rules are applied according to Figure 1-2 on Page 3. The flowchart rules are not defensible, especially in regard to detection frequency in sediment (step 3) and in relation to media defined as "SW, TZW, clam/crayfish/sculpin/SMB tissue". For these media, an individual sample must have an HQ >5 and a log Kow >4 to be carried forward as a COPC. Once it is detected in tissue, the risk should be assessed in the risk assessment regardless of the HQ, and it doesn't matter if it has a log Kow>4.
   a. Refined Sediment Screen
      i. Table 2-4, Refined Sediment Screening: Additional chemicals are screened out in this stage include Diethyl phthalate, Dimethyl phthalate, 1,3,-Dichlorobenzene, and heptachlor.
   b. Refined Screen for Invertebrate Tissue Residue
      i. Table 3-7 Refined Screening for Field Collected Clam Tissue:
      ii. I am not sure how Dimethyl phthalate screens out as it is unclear what the detection limit was and what the "no data" footnote means. If it was higher than the benchmark it should be retained.
      iii. Table 3-8 Refined Screening for Crayfish Tissue

iv. The footnote indicates “ND” equals “no data”, but it must indicate “non-detect”. However, detection limits are well above benchmark, so the contaminants screened out here should be carried forward as BERA COPCs including dibutyl phthalate, diethyl phthalate, and dimethyl phthalate.
v. Table 3-9 Refined Screen for Multiplate Invertebrates
vi. Table 3-10 Refined Screen for Mussel Tissue: Everything is retained as a COPC, biggest problem is missing COIs from the SLERA step.
vii. Table 3-11 Refined Screen for Laboratory Clam Tissue: Everything is retained as a COPC, biggest problem is missing COIs from the SLERA step.
viii. Steady state tissue concentrations presented here need to be reviewed
ix. Table 3-12 Refined Screen for Laboratory Worm Tissue: Everything retained as a COPC, biggest problem is missing COIs from the SLERA step.
x. Steady state tissue concentrations presented here need to be reviewed

c. <u>Refined Screen For Fish Tissue Residue</u>
i. Table 3-13 Refined Screen for Largescale Sucker
ii. “ND” for not detected is shown for dibutyl phthalate, Diethylphthalte, Endrin, alpha-HCH, beta HCH and delta HCH so these contaminants are screened out. The detection limits for several of these contaminants are higher than the benchmark, but they are still not considered BERA COPCs. For example, the detection limit of diethylpohthalate was 520 ppb compared to the benchmark of 220 ppb. The same is true for Endrin with a non-detect concentration of 31 ppb and a benchmark of 25. This also occurred for the lindane isomers and dibutyl phthalate. These need to be retained as COPCs as discussed as a data gap.
iii. Table 3-14 Refined Screen for Juvenile Sturgeon Tissue: Everything retained as a COPC, biggest problem is missing COIs from the SLERA step.
iv. Table 3-15 Refined Screen for Juvenile Chinook Salmon Tissue:
v. BEHP and Diethyl phthalate (detection limit 1,300 ppb; benchmark 220 ppb) screened out on the basis of “ND” when detection limits are higher than benchmark. These should be carried forward as BERA COPC.
vi. Table 3-16 Refined Screen for Peamouth: Everything retained as a COPC, biggest problem is missing COIs from the SLERA step.
vii. Table 3-17 Refined Screen for Sculpin
viii. Screened out dibutyl phthalate when detection limit is higher than benchmark.
ix. Table 3-18 Refined Screen for Smallmouth Bass
x. Screened out delta-HCH due to benchmark lower than detection limits.
xi. Table 3-19 Refined Screen for Northern Pikeminnow
xii. Screed out alpha-HCH, beta HCH, and delta HCH but detection limits were higher than benchmark.
xiii. Table 3-20 Refined Screen for Lamprey Ammocoete Tissue:
xiv. Screened out Diethyl phthalate but detection limits were higher than benchmark.

d. Refined Dietary Screen for Fish: Tables 4-13 through 4-19. These tables are not useful until the appropriate acceptable tissue concentrations are determined. However, it doesn't appear additional COIs were screened out during this step.
e. Refined Dietary Screen for Wildlife: Tables 4-32 through 4-38. Only one additional chemical screens out here and it is for Osprey and dibutyl phthalate. It appears that from the SLERA screening for osprey (Table 4-30), the maximum detected concentration in tissue went from 660 U to 37 T ug/kg. **It appears the biggest change from the SLERA to the refined screen is that elevated non-detect concentrations are dropped, and chemicals are screened out on this basis.** These chemicals should be carried forward in the risk assessment, and discussed in terms of data gaps and uncertainly.
f. Refined Surface Water Screen
g. Refined TZW Screen, Table 6-5:
    i. Total Selenium and styrene are dropped out based by dropping elevated detection limits.
h. Background Comparison Tables 7-1 (sediment) and 7-2 (surface water). The background dataset used for this analysis needs to be reviewed. What is defined as background? I guess Section 7.0 of the DRAFT RI is needed to complete this review. See also Attachment 11, "Evaluation of Background and Upriver River Reach Concentrations".

i. Fish Individual Sample and Dietary Component Assessment, Attachment 12: This assessment builds from the SLERA and refined screen. Therefore, comments on this section will be limited until it is shown that all COPCs have been properly identified.

3. **SECTION 6.0, BENTHIC INVERTEBRATE RISK ASSESSMENT (Main Text)**
    a. Table 6-1: *Lumbriculus* (laboratory worms) are missing from this table showing benthic invertebrate receptors.
    b. Section 6.0, Page 117: The text states "none of the generic SQGs could reliably predict toxicity in Portland Harbor sediment, therefore the generic SQGs were not used in risk characterization." This depends on an agreed upon definition of "reliability" which still needs to be determined. I did not review Attachment 7 in detail, however, the selection of reliability criteria for low SQGs is inaccurate because the expected false positives is set too high for these values (LWG 20%,, McDonald cited 50% on our call, DEQ would say focus on false negatives and do the best you can at reducing false positives). The LWG reliability requirements are presented on page 138 (both false negatives and false positives should be below 20%, and overall reliability should be above 80%), and are based on the draft Washington State freshwater guidelines (Avocet, 2003). It is not a function of low SQGs (e.g. TECs) to predict toxicity accurately, as they predict sediment where there would be no toxicity. If a value is above a low SQG, further bioassay testing should be completed to determine toxicity. This is also important since these criteria were used to evaluate the logistic regression, and is cited for the reason this line of evidence was not included (page 129).

c. Table 6-7, Page 132, Chemicals Screened Out Prior to Model Development; These chemicals may be important toxicity drivers in localized areas of the site. There may not be enough information to include them in a site-wide predictive mode, but AETs (highest concentration with No Effect) should be presented for these chemicals.
d. Table 6-8, Page 136: The ANOVA analysis will need to be reviewed to agree with the list included in this table. Several were screened out on this basis here that were included in the RSET model. I don't see a presentation of this information in this report. Also, the change in control normalization and biomass endpoint could significantly change the conclusions here for some chemicals.
e. Section 6.1, Page 119: The footnote states "since the toxicity tests were conducted, locations where 13 of the 269 surface sediment samples had been collected have been dredged. These 13 samples were therefore excluded from the toxicity assessment". Were these eliminations appropriate? Was then used to represent conditions in these areas? Were new sediment concentrations collected here or was it assumed that the areas are now clean a no exposure point concentration is used? Several of these samples exceeded high thresholds (Page 124, footnote "e" in Table 6-3).
    i. Further details here were not reviewed until the changes EPA requested are made and a new benthic model is submitted.
f. Section 6.1.2.1, Page 137, Toxicity Assessment Based on Bivalve Growth and Mortality: This evaluation is new, and the raw data should be submitted (it was not submitted in previous documents). These data should be included as a line of evidence in the final risk characterization.
g. Section 6.2.3, SQG Derivation: See above comment on reliability. However, calculating all error and reliability rates for each set of the initial SQGs by using the pooled endpoint (results shown in Table 6-9) will likely lead to inappropriately reduced reliability. ***The criteria for removing a chemical from the model dataset were inappropriate and likely led to a reduced set of SQGs to (Table 6-10)***. SQG values for chemicals removed after model development removes significant information in the weight of evidence framework and ***implies that there is no toxicity associated with the chemical***, which is inaccurate. Chemicals that were shown to correlate with toxicity were removed based on criteria outlined in bullet 4, Page 139. Further comment and review will be needed after the model is submitted.
h. Section 6.2.4.1, Page 141, Comparison of Study Area Concentrations to Site-Specific SQGs: ***Comparison to SQGs should be presented here regardless of reliability.*** Instead, only those that were determined to "reliably predict toxicity were evaluated (see comment on Section 6.0, Page 117). What does the statement "toxicity could not be assessed at 37 locations because chemical analyses were not performed for the site-specific SQGs"?

4. **TRANSITION ZONE WATER – BENTHOS:** Table 6-26 showing the TRVs for TZW COIs is a reduced table from Table 6-2 (Attachment 5) that shows the complete list of COIs and TRVs used to

reduce down to this list. This more complete list and associated TRVs need to be reviewed. (a few examples below).

i. Table 6-27: This table leaves off important COIs such as dioxins and furans.
ii. Section 6.6.5: This section is highly biased toward the "benthic organisms have mechanisms to reduce / eliminate exposure" speaking to the ecological relevance of the findings. The other side needs to be presented.
iii. Section 6.6.6, Recalculation of the DDT AWQC: The DDT water TRV was recalculated here to "be more representative of direct exposure and toxicity to aquatic organisms" needs to be reviewed. The new value is of 0.011 ug/L, and they go on to make the statement "the toxicity benchmarks (i.e AWQC) originally selected for DDx compounds in water are not ecologically relevant for evaluation of risks to benthic invertebrates, fish, or plants". I don't think we should be using a new DDT number to evaluate effects.

5. **SECTION 6.7, BENTHIC RISK CONCLUSIONS:** I am not sure what they mean by " the measurement endpoints are determined at the organism level" and "conclusions about unacceptable risk to populations and communities can by drawn only by extrapolating from potential effects in individual organisms". Risk was measured by using ***test populations*** (e.g. laboratory test populations of *Hyalella* and *Chironomus*) to infer risk to site populations. Effects on these populations are used to infer risk about site communities (e.g. changes in growth, mortality has been linked extensively to changes in benthic community structure (e.g. EPA 2000). For example, changes in growth in bioassay tests have been linked to changes in community structure and diversity. Organisms that do not grow properly cannot emerge from sediment to reproduce. Ability to colonization new substrate is also affected. For the benthic invertebrate tissue residue lines of evidence, effects and risk was measured on ***tissue composites***, not individuals. The only way to truly evaluate changes in community structure is to measure environmental degradation by evaluating alterations in benthic community structure in the field. Since this evaluation was not done on the site, ***it must be assumed that changes in endpoints such as growth and mortality results in benthic community effects in the field***. It is further stated that "localized TRV exceedances do not indicate population- or community-level risks". This statement is not accurate. Rather, ***single exceedances represent population level effects and degradation of the benthic community degradation in that localized area.***

   a. <u>Weight of Evidence (also section 6.7):</u> It is determined here that the predictive models represent a stronger line of evidence that empirical data because "the historical distribution of chemicals in sediment is limited, and sediment samples do not integrate well of a wide area". Mapping and use of the food web model was then used to predict where exceedances would occur, and identify potential risk areas (PBRAs). This is not an appropriate use of the weight of evidence approach, as the models do not incorporate empirical results from the site. Each line of evidence should be overlaid on a map.

i. The rest of this section was not reviewed any further until issues related to an updated benthic model and agreement on how lines of evidence will be presented and used in the risk assessment is made. However, only two lines of evidence were ultimately evaluated (site specific SQGs and exceedance of tissue residue TRVs). Tables 6-30 through 6-32 should show all lines of evidence outlined in the problem formulation including:
    1. Empirical toxicity testing results
    2. Logistic Regression Model Exceedances (not just the Floating Percentile Model)
    3. Water exposure to TRVs including surface water and transition zone water
    4. Bulk sediment contaminant concentrations compared to sediment quality guidelines
        a. Consensus Based SQGs (TECs / PECs and related quotients)
        b. Mechanistic-based SQGs (Equilibrium Partitioning)
        c. Empirical SQGs (PELs / TELs, ERLs / ERMs, AETs, LRM, and related quotients.

ii. These tables are also highly biased in the "risk conclusions sections" almost in every case concluding "negligible risks to the benthic community" based on their WOE approach.

b. Section 6.7.2.4: SPI imaging is not an effective tool for determining community health. Stage 1, 2, and 3 are not relatable to diversity and abundance, and do not characterize the epibenthic community where a large proportion of the benthic community resides. We are not just protecting the oligochaete and polychaet (worms) of the benthic community. In fact, a preponderance of these kinds of organisms shows a benthic community that is significantly degraded. This evaluation does not support the statement "the data suggest that the physical environment in the Study area can explain the condition of the benthic community throughout most parts of this area of the river".

6. **SECTION 7.1 FISH TISSUE RESIDUE ASSESSMENT:** See also comments on the SLERA and refined screens.
    a. Section 7.0, Page 237: These are not estimates based in individuals – they are based on composites of many fish for different locations from the site. The TRVs are based on laboratory populations measuring effects. The language here about conservatism is unsupported unless population attributes will be measured in the future.
    b. Section 7.0, Page 238: Not mentioned here is the ***high degree of uncertainty associated with "information about feeding rates, foraging areas, prey home ranges, and diets" for fish species making it a particularly weak line of evidence as presented here***.
    c. Section 7.1, Page 239: EPA's problem formulation was not followed in this case. This will have to be reviewed and possibly re-done.

d. Section 7.11, Page 242, Carp: It was my understanding that carp concentrations and risk were to be presented to support the uncertainty section and to ensure we were being protective of carp by using other fish species as surrogates.
e. Section 7.1.2.1, Page 243, Step 1, Individual sample-by-sample tissue-residue conc: It should be noted that ***Attachment 4 only shows EPCs for those chemicals that screened in and were identified as final COPCs***. This makes it very difficult to evaluate other chemicals either to evaluate changes in TRVs, or to evaluate uncertainty.
f. Section 7.1.2.1, Page 243, Step 2: ***Step 2 is not consistent with EPA's problem formulation which stated "evaluate on a composite by composite basis to protect for localized population effects independent of fish homerange".***
    i. Calculation of 95th UCL on the mean may be appropriate to evaluate species foraging on fish tissue (e.g. osprey, mink), the appropriateness to evaluate risk to fish populations is tenuous. These samples are already composites (averages) of tissue residue concentrations over a given area. ***Each composite should be evaluated separately (Step 1) as the final risk characterization step***. The 95% UCL on the mean values used for fish tissue residue for this step are not presented, so it is impossible to compare risk results from Step 1 and Step 2. Step 1, presented in Attachment 12 (Table 2-1) should be used for risk characterization. This process should include a complete list of COPCs based on previous comments (e.g. dioxins and furan / PCB TEQ). The locations of these individual fish tissue samples should be shown and mapped.
    ii. EPA's problem formulation also indicated that if possible the true concentration range from the composites should be presented.
g. Section 7.1.2.1, Assessment Based on Individual Samples: Attributes to do a population level risk assessment were not made, including concentration determinations by age class, individual concentration measurements, fecundity, etc.
h. Section 7.1.2.2, Predicted Tissue EPCs: BSAFs (calculated where they did not) and BSARs should be used to validate the model predictions for sculpin. The food web model is a site wide model, predicting average tissue concentrations for this species. It is unclear if these predictions produce accurate estimates of risk to this species.
i. Section 7.1.2.2, Predictions of Dioxin / Furan Concentrations: The footnote states **"dioxins and furans were not tissue COPCs for sculpin (or any fish receptor), so the mechanistic model was not used to predict dioxin and furan sculpin tissue concentrations"**. While I do not agree with their conclusions on COPC determinations, either way the objective here was to predict tissue where tissue was not collected. Therefore, this statement does not apply – they should have been predicted and screened. The predictions they did make are extremely limited – only Total PCBs, beta-HCH, and Total DDX.
j. Table 7-5, Selected Fish Whole-Body Tissue TRVs: I am assuming this has been reviewed by EPA, but this only represents the TRVs for the "final selected COPCs". See also the larger list of tissue TRVs presented in Attachment 5 Table 3.1 that need to be reviewed.

i. DDT: This TRV should be reviewed, as it did not include egg or embryo residues in the SSD. This TRV makes a difference for some tissue (e.g. smallmouth bass, see below).
ii. PCBs: Same thing as DDT – why weren't studies reporting adverse effects associated with egg or embryo residues included in the SSD?
iii. TEQ: No dioxin furan TEQ or total TEQ was calculated, and studies showing effects to these congeners were excluded from TRV development of TEQ effects. Therefore, it is unclear if the total PCB TRV is protective of dioxin like effects – esp. at 930 ug/kg. If an appropriate fish TRV were used (e.g. 40-50 pg/g), more fish samples would be identified.

k. Section 7.1.3, Page 249, 5th and 10th Percentile: The selection of the 5th and 10th percentile for protection of threatened or endangered and populations, respectively, should be supported. The DDT number of 1.6 for populations should be revisited, as it did not include egg or embryo residues in the SSD. This number is very close to the maximum smallmouth bass concentration of 1.5 mg/kg, which is screened out, not evaluating total DDT any further for this receptor.
l. Section 7.1.4.2.1, Large-Home Range Fish, Table 7-8: This table should present individual composite risk, not using at 95% UCL concentration – this is only shown in Attachment 12. This table should be similar to Table 7-10 for smallmouth bass. HQs for carp would be significantly higher for Total PCBs. Large home range fish such as largescale sucker, peamouth and pacific lamprey ammocoetes had the following changes from the SLERA and refined screen"
    i. Largescale Sucker: Aluminum (max 154 mg/kg), chromium (2.77 mg/kg), BEHP (3 mg/kg), 4,4'-DDD (0.15 mg/kg) and Total DDX (0.67 mg/kg) were all dropped in the final risk characterization step. ***This process drops site wide risk drivers from the risk characterization process. Maps presented on a composite sample-by sample basis are only for the refined list.***
    ii. Peamouth: Aluminum was dropped due to uncertainty in the TRV (max detect 185 mg/kg).
    iii. Lamprey ammocoetes: Aluminum (max detect 281 mg/kg), diethyl phthalate, and 4,4'-DDD (max detect 0.0547 mg/kg) were all dropped and not discussed here. These were due to uncertainties in the TRV, high detection limits, and changes in the TRV, respectively.
m. Section 7.1.4.2.2, Table 7-9, Sculpin: Several COPCs are dropped from the SLERA and refined screening including aluminum, BEHP, 4,4'-DDD, 4,4,'-DDT, and beta-HCH. Aluminum and BEHP were dropped because of "uncertainty with the TRV" (instead, discuss in uncertainty section), even though there were significant exceedances of both the TRV used in the SLERA and refined screen (0.39 mg/kg) and the final TRV (9.6 mg/kg) (max detect was 28 mg/kg). 4,4'-DDD was dropped because the TRV went from 0.054 mg/kg to 1.6 mg/kg total DDX TRV (highest detect 4,4'-DDD was 0.305 mg/kg), 4,4,'-DDT was dropped because the TRV went from 1.7 mg/kg to 1.6 mg/kg Total DDX TRV

(highest detect 4,4'-DDT 1.7 mg/kg). These congeners should have been carried forward, not just total DDx. Beta-HCH was dropped because TRV went from 0.0049 mg/kg to 0.20 mg/kg (highest detect was 0.0062 mg/kg). ***This process drops localized effects from the risk characterization process. Maps presented on a composite sample-by sample basis are only for the refined list*****,** which is misleading.

n. Section 7.1.4.2.2, Table 7-10, Smallmouth Bass: This table drops BEHP, which had 2 exceedances of the TRV at 87 mg/kg and 32 mg/kg. Table 7-7 (page 254) also show these exeedances. Therefore, it is unclear why Table 7-10 does not show the BEHP as "area specific tissue 10th percentile LOAEL HQ". This COPC is dropped due to "uncertainty in the TRV". This is not appropriate. Antimony, Cadmium and Lead are dropped because the maximum detected concentrations were higher than the TRV (Table 3-18, Attachment 5). Total DDx and 4,4'-DDD were also dropped in this step because the TRV went from 720 mg/kg to 1,600 mg/kg from the screening steps to final selected TRVs. Antimony and Cadmium also screen out due to changes in the TRV (antimony went from 0.03 mg/kg to 9.0 mg/kg and cadmium went from 0.09 mg/kg to 0.22 mg/kg). Were these changes approved by EPA? Retaining of appropriate TRVs is important in identifying localized effects. Antimony is significantly higher than all other samples in the harbor off the Goldendale Aluminum facility, with a concentration of 7.6 mg/kg. The cadmium maximum was also found in this area with a high of 0.20 mg/kg. ***This process drops localized effects from the risk characterization process. Maps presented on a composite sample-by sample basis are only for the refined list.***

o. Section, 7.1.4.2.2, Table 7-11, Northern Pikeminnow: Dropped from the SLERA and Refined Screen was Total DDx (max detect 1.9 mg/kg) due to the change in TRV from 0.29 mg/kg to 1.6 mg/kg. ***This process drops a site wide risk driver from the risk characterization process. Maps presented on a composite sample-by sample basis are only for the refined list.***

p. Section 7.1.4.3.1, Tissue Data from the Downstream and Downtown Reaches: Data from downstream of the study area do exceed TRVs and it was inappropriate not to include these samples in the risk assessment. Since these samples were not carried through the risk screening process, it is impossible to see the COPCs that would have been identified. The "CDF approach" (figures 7-3, 7-4 and 7-5) does not represent a risk screening. One example includes sculpin exceedances of copper at 3.1 mg/kg (east bank, 3rd highest in the river).

q. Section 7.1.4. 3.3., Tissue Data from the Upriver Reach: The fish ***collected upstream were significantly larger than the fish collected from the harbor.*** Larger fish in the harbor were "thrown back" and not analyzed. Therefore, this comparison is not comparing like fish size and there is no basis in the conclusions.

r. Section 7.1.4.3.3, Additional Regional Data: It is inappropriate to compare average values only.

s. Section 7.1.4.4, Table 7-13, Page 267-269; This table presents a very skewed version of uncertainty.

t. Section 7.1.4.5, Evaluation of Non-Target Receptors: This process should have occurred for carp as well.

7. **SECTION 7.0 FISH DIETARY ASSESSMENT:** I did not review this section in this review cycle. Attachment 13, Details on Exposure and Effects Assumptions for the Fish Dietary LOE also needs to be reviewed. See SLERA and Refined Screen comments.
    a. Fish Dietary: Sample by sample HQs are based EPCs based on UCLs on prey and sediment samples (footnote, Attachment 12, page 6). As per EPA problem formulation, individual prey samples were to be screened individually to reduce uncertainty in determining a priori fish "relevant exposure areas".

8. **SECTION 7.3 SURFACE WATER ASSESSMENT:**
    a. Section 7.3, Page 315: ***It is not appropriate to calculate 95% UCL on water concentrations for comparison to larger home range fish.*** Even if they are wider ranging they will still be exposed above chronic or acute TRVs during some time-frame. All fish except sculpin were evaluated as 95% UCL on the mean over some exposure area. For all practical purposes all fish should be evaluated on a sample by sample basis. ***As per the problem formulation "compare every individual water sample to water TRVs. Consider exceedance of acute or chronic values at any scale a risk (near bottom and integrated) due to lack of sufficient samples to accurately obtain better exposure resolution".*** Therefore, the assessment for sculpin, with the addition of the peristaltic samples, should be used to assess risk to all fish. This is presented in the text, but not in Table 7-36, where only 1 mile exposure areas are presented, but see Maps 6-30 through 6-34. There are several widespread exceedances – esp. of DDx and isomers, that supports the conclusion that these compounds present a site wide risk to fish receptors, contrary to the conclusions make in the tissue residue section.
        i. While inappropriate, the 95% UCL values used in the risk assessment along with distribution types, and Pro UCL recommended UCLs are not presented here or in Attachment 4 as stated, making it impossible to see how conclusions would change. While 95% UCLs may be presented somewhere in the document, I have been unable to locate them.
    b. Section 7.3.2.1, Page 318: ***It is not appropriate to drop the results from the peristaltic samples*** where XAD was collected in the same area. Just because the XAD is based in high resolution doesn't mean it represents the same exposure point concentration in terms of spatial and temporal distribution, nor does it represent the same filter size for evaluating total and dissolved metals (XAD was a bigger filter size).
    c. Section 7.3.3.1, Page 320: ***Final Water TRVs and risk characterization (beyond SLERA and Refined Screen) are based on a re-evaluate of water criteria for total PCBs and 4,4'-DDT (total DDx).*** This needs to be reviewed.
    d. Section 7.3.3.2, page 322: Have the LC50 values for lamprey been approved or reviewed by EPA?

e. Section 7.3.4, Risk Characterization: This section should be re-done incorporating the comments above. 95% UCLs on the mean and alternative TRVs should not be used.
f. Section 7.3.4.5, Summary of Surface Water COPCs: while 11 COCs were identified, 19 were originally identified, and several were dropped due to no available TRV (Table 5-3, Attachment 5). This needs to be reviewed in more detail.

9. **SECTION 7.4, ASSESSMENT OF BENTHIC FISH HEALTH AND PAH EXPOSURE:** I did not review this part of the review cycle.

10. **SECTION 7.5, FISH TZW ASSESSMENT**: ***Results of the TZW assessment should have been incorporated into the risk conclusions section***

11. **SECTION 7.6, FISH RISK CONCLUSIONS AND UNCERTAINTY ANALYSIS:**

a. Table 7-39: This table needs to be revised.
b. Section 7.6.2, WOE Evaluation: When the correct risk characterization is completed and lines of evidence dropped are brought back in, this will actually be useful. At a first cut, it shows that contrary to their conclusions, DDx and total TRVs are site wide risk drivers and present a risk to fish populations.
c. Section 7.6.2, Page 350: Until population data such as "life stage class abundance and maternal PCB concentration data are collected for this site" there is no basis to conclude no population risk.
d. Tables 7-40: Not reviewed in this part of the review cycle. Better done when comments incorporated on previous sections.

12. **SECTION 8.0, WILDIFE RISK ASSESSMENT:**

a. Section 8.1.1, Table 8-2, COIs Not Evaluated: There are TRVs for several of these COIs. For example, silver has EPA Eco SSL TRVs and others…
b. Section 8.1.2.1, Exposure Concentrations:
    i. Step 1, EPCs by individual prey composite tissue and sediment concentrations, Attachment 17: ***Risk characterization should be based on Step 1.*** Comments to follow on Attachment 17.
    ii. Step 2, EPCs be receptor-specific exposure areas, Attachment 16: Exposure area assumptions do not follow problem formulation.
        1. ***Shorebird EPCs based on 2-mile 95% UCL increment exposure areas for COPC concentrations***. The problem formulation outlined a beach by beach risk characterization. Justification was "sandpipers represent all omnivourous / invertivorous guilds on the site (including cliff swallows, riparian songbirds, smaller ranged species). Individual beach scenario is most sensitive evaluation because the primary forage area could be one beach, and developing young may only forage along one beach."

2. ***For hooded merganser, bald eagle, osprey and mink exposure based on 95% UCL of 1-mile increments of the study area.*** Important concentration areas are bifurcated by this analysis. EPA outlined a similar RBC (TSC) approach to evaluate the merganser, bald eagle, and osprey.
    a. Sculpin and smallmouth bass also on 1-mile 95% UCLs.
    b. Should be a 1 mile exposure, but individual prey should have been screened using appropriate TSC, and ***"identify dietary components associated with greatest risk within 1-mile segments (progressed as ½ mile increments)."*** Instead, dietary components (carp, juvenile Chinook salmon, largescale sucker, and peamouth) were based on 95% UCL of the study area concentrations and a forward calculation was done.
3. ***River otter based EPC on 3 mile 95% UCL.*** Important areas of the site bifurcated. Direction was to "evaluate EPOC within 3-mile segments (progressed as 1.5-mile increments so that a high contamination area is not bisected." This is exactly what happens by bisecting at RM 7.5, where the highest Total TEQ can be found. Also, why are the increments including RM 1.5 and above RM 10.5 in the 3 mile segments? These are heavily including non-initial study area segments, and concentrating the evaluation into two major segments - one major 3 mile segment from 4.5 to 7.5 and 7.5 to 10.5.

iii. As per Page 373, Attachment 4 does not show UCL concentrations. Only detection frequency, min detection, max detection, mean detection and range presented.

iv. <u>Step 3</u> misses the whole point of what we outlined in the problem formulation. No probabalisic or uncertainty analysis is used to evaluate the assumptions about dietary composition, one of the larger areas of uncertainty we were attempting to solve.

c. Section 8.1.2.2.1, Dietary Dose Approach, Equation 8-3: The "SUF" should be removed from the equation, as the problem formulation directed all to be evaluated as using the site year round. If a SUF is to be used, it would not be in the denominator, but a factor applied to the after TRV/FIR/BW. Was a factor different from 1 used in this equation?

d. Section 8.1.2.3.1, Table 8-4, Dietary-Dose Exposure Parameters: This table needs to be reviewed.

e. Section 8.1.2.3.2, Page 379, Bird Egg Exposure Parameters, BMFs: Based on no BSAR relationship found by the LWG using Willamette River data, the conclusion here is that the BMF approach is unreliable except for total TEQ, and later is dismissed entirely as a line of evidence. The "factor" approach is defensible compared to the "regression" approach. This is the same argument used to dismiss relationships between sediment and tissue. The data and analysis used to calculate the BMRs should be submitted for review, but I would recommend using the BMF approach outlined previously in this

project. The BMF of 10 should be used instead of the BMR presented here. Ultimately, osprey egg concentration data will be available to confirm this relationship.

f. Section 8.1.2.3.3, Page 384-385:
    i. Table 8-7: Prey consumption: Smallmouth bass should be evaluated with the bald eagle.
    ii. Table 8-8: These prey portions are not supportable. The use of this methodology was addressed by EPA's problem formulation, which stated "vary prey items probabilistically to identify components associated with the greatest risk within 1-mile segments (progressed as ½ mile increments)".

g. Section 8.1.2.4, Data Used to Derive Sandpiper Exposure Concentrations: BSAFs should have been calculated for contaminants modeled using the mechanistic model. This is an important check that the model is adequately predicting concentrations on a local scale. Also, BSAF models for invertebrates closely tied to the sediment may be more predictive and accurate than a food web model. The criteria for developing BSARs was too restrictive for developing relationships (significantly positive slope at a p of 0.05 and an r squared greater than 0.030.
    i. Table 8-9, Page 387: Several of these beach areas do not match up with shorebird sampling areas. Several addition areas can provide estimates where "none" is listed (e.g. LWG 004 at beach area B4). ***Simply not estimating exposure to shorebirds at all in these areas is unacceptable.***

h. Section 8.1.2.4, Table 8-10 refers to the results of the mechanistic model for predicted shorebird prey concentrations. Tables with the predicted concentrations from the mechanistic model are presented in Table 3-7 from Attachment 3. However, concentrations are only presented for tributyltin ion, Total PCBs and Total DDX. Values for Dioxin/ Furan TEQ, Total TEQ, Aldrin, and Sum DDE, listed in this table as COPCs, are not presented. Were predicted concentrations not calculated for these chemicals? Only average concentrations are presented, with an associated range of values. A table should be presented to show the predicted concentrations for each sample used in the EPC calculations for all COIs. Since BSAFs were not calculated for modeled contaminants such as PCBs, pesticides, etc., this information is needed to ensure modeled results are lining up with expected concentrations from other lines of evidence.

i. Section 8.1.3.1.1.:
    i. Table 8-11, Bird Dietary Dose TRVs: The list of TRVs for birds needs to be reviewed (see comments on the screening table 4-24 in Attachment 5). Some There are some bird TRVs where EPA Eco SSLs were adjusted to calculate a LOAEL. However, this was not done in all cases (e.g. Total DDT LOAEL was derived from the same study as the NOAEL). See previous dioxin / furan comment.
    ii. Table 8-12, Mammalian Dietary Dose TRVs: The list of TRVs for birds needs to be reviewed (see comments on the screening table 4-25 in Attachment 5) for complete list.

j. Section 8.1.4.2.1, Pages 410-415, Spotted Sandpiper: Risk characterization based on 2-mile beach assessment for Spotted Sandpiper – I did not review these sections. Concentrate on results from Attachment 17.
    i. ***Steady State Adjustment to worm EPC needs to be reviewed (Attachment 3, as this is a big component of the sandpiper diet.***
k. Section 8.1.4.2.2, Hooded Merganser, Pages 416-417: Did not review, 95% UCLs could not be verified, increments should be at ½ mile.
l. Sections 8.1.4.2.3 through 8.2.3.2.3 and Pages 417-473: Did not review the rest of the wildlife section.

13. **SECTION 9.0, 9.1, 9.2, AMPHIBIAN RISK ASSESSMENT:** Not reviewed during this part of the review cycle. This section should follow closely with the fish and invertebrate identification of COCs.
    a. ***Alternative water quality criteria for DDx (0.011 ug/L) and total PCBs (0.19 ug/L) were used to determine risk conclusions for amphibians.*** Table 9-1 shows exposure areas with HQs >1.0, but using alternative water quality criteria. Additional DDx isomers are dropped out. Why?
    b. Sampling Locations included: Why are several transect samples included (e.g. W011, W023, and W025W and E from Round 3)? Where is W05?

14. **SECTION 10.0, AQUATIC PLANT RISK ASSESSMENT:** Not reviewed during this part of the review cycle.

15. **SECTION 11.0, ECOLOGICAL RISK CONCLUSIONS:** Not reviewed during this part of the review cycle (brief comments provided).

    a. Section 11.3, Page 515 and Table 11-2: ***Additional risk characterization / management conclusions are made by determining low magnitude HQs, frequency and extent pose "no unacceptable risk". All lines of evidence for benthic invertebrates, fish, wildlife receptors, amphibians and plants are dropped or "unknown". The only pathways for which unacceptable risk is identified is for Total PCBs and benthic invertebrates, birds, and mammals), Total TEQ for mammals, and DDx for invertebrates. It is stated that "other COCs were found not to pose unacceptable risk to the ecological assessment endpoints assessed in the BERA". This is a flawed reduction in COCs by scale and different receptor groups.***